bp

George E. Tacquard
Business Unit Leader,
Nitriles

BP America Inc
Mail Code N-1
150 West Warrenville Road
Naperville, IL 60563-8460

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June 28, 2002

The Honorable Christine Todd Whitman
Administrator
United States Environmental Protection Agency
1200 Pennsylvania Avenue, N. W.
Washington, D.C. 20460

Re: Petition to Delete Acetonitrile from the List of Toxic Chemicals at 40 C.F.R. § 372.65

Direct: 630 961 7968
Fax: 630 961 7664
tacquage@bp.com

Dear Governor Whitman:

BP Chemicals Inc. ("BP") hereby petitions the United States Environmental Protection Agency ("EPA") to remove acetonitrile (Chemical Abstracts Service Number 75-05-8) from the list of Toxic Chemicals at 40 C.F.R. § 372.65 that are subject to the reporting requirements of the Toxic Release Inventory ("TRI"). This BP petition is filed under section 313(d)(3) of the Emergency Planning and Community Right to Know Act of 1986 ("EPCRA"). It builds upon a previous petition filed in 1998 by BP and the GNI Chemical Company ("GNICC") ("the 1998 Petition"). That petition was denied by EPA, but only after determining that BP and GNICC had satisfied some, but not all of the requirements for demonstrating that acetonitrile should be deleted from the TRI list of toxic chemicals. This petition builds upon the 1998 Petition, EPA's response to that petition, and subsequent BP demonstrations and EPA determinations about the status of acetonitrile under the delisting criteria of EPCRA section 313.

The enclosed petition demonstrates that acetonitrile meets all of the section 313 criteria for delisting. First, acetonitrile is not known to cause and cannot be reasonably anticipated to cause significant adverse human health effects at concentrations that are reasonably likely to exist beyond facility boundaries as a result of continuous or frequently recurring releases. EPA determined this to be true when it acted on the previous 1998 Petition. There is no new evidence since 1999 that provides any basis for changing that assessment, and the information on releases shows an overall reduction rather than any increase in the release levels for acetonitrile.

Second, at exposures likely to be found beyond facility fencelines, acetonitrile is not known to cause and cannot be reasonably anticipated to cause cancer or teratogenic effects or serious irreversible reproductive dysfunction, neurological disorders, heritable genetic mutations, or other chronic health effects. EPA has established a reference concentration (RfC) for acetonitrile through the Integrated Risk Information System (IRIS) specifically based on the mortality evidence that has concerned EPA. The RfC establishes a safe level for lifetime exposure to acetonitrile, and the exposure studies for acetonitrile show that exposures will not occur at levels

above the RfC. Accordingly, generally accepted scientific principles require EPA to accept the results of its own IRIS assessment and to delete acetonitrile from the TRI list of toxic chemicals.

Finally, acetonitrile is not known to cause or reasonably likely to cause significant adverse effects to the environment because it is not toxic or persistent and does not readily bioaccumulate. The only concern that EPA expressed in the past related to the possible contributions of acetonitrile to ozone formation because it could be viewed as a volatile organic compound (VOC). EPA's Office of Air Quality Planning and Standards (OAQPS) has determined, however, that acetonitrile is not sufficiently photochemically reactive to contribute significantly to ozone formation in the ambient air. Accordingly, EPA should issue a coordinated determination deleting acetonitrile from the TRI list and from the definition of VOC under the Clean Air Act regulations. BP reiterates its requests for both actions in this petition.

Because acetonitrile meets all three of the EPCRA criteria for delisting, it should be removed from the list of Toxic Chemicals at 40 C.F.R. § 372.65.

If you or your staff have any questions about this petition, please contact Robert Van Voorhees at (202) 508-6014 at the law firm of Bryan Cave LLP. Thank you very much for your consideration of this Petition.

Respectfully submitted,

George E. Tacquard

George E. Tacquard

Enclosures

cc: Kimberly Nelson, Assistant Administrator for Environmental Information

PETITION OF BP CHEMICALS INC.

TO DELETE ACETONITRILE

FROM THE TRI LIST OF TOXIC CHEMICALS

Robert F. Van Voorhees
Jill M. Zucker

Bryan Cave LLP
700 Thirteenth Street, N.W.
Suite 700
Washington, DC 20005-3960
(202) 508-6000
(202) 508-6200 FAX

Counsel to BP Chemicals Inc.

Dale E. Strother, DABT

BP Chemicals Inc.
1300 Wilson Blvd.
Suite 1200
Arlington, VA 22209
(703) 741-5020
(703) 741-5110 FAX

PETITION OF BP CHEMICALS INC. TO DELETE ACETONITRILE FROM THE TRI LIST OF TOXIC CHEMICALS

EXECUTIVE SUMMARY

BP Chemicals Inc. ("BP") petitions the United States Environmental Protection Agency ("EPA") to remove acetonitrile from the list of toxic chemicals subject to the reporting requirements for the Toxics Release Inventory ("TRI"). That list is maintained by EPA, and this petition is filed, under Section 313 of the Emergency Planning and Community Right-To-Know Act of 1986 ("EPCRA").[1/]

This is BP's second petition to delist acetonitrile from the TRI list of toxic chemicals. BP and The GNI Chemical Company first petitioned EPA in February 1998. After finding that BP had met many of the demonstration requirements for delisting, EPA denied that petition on February 24, 1999.

There are three essential requirements that must be met to delist a chemical from the TRI list of toxic chemicals. First, the Petitioner must demonstrate that there will be no acute human health effects from exposure to the chemical at concentrations that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases. Second, the Petitioner must show that the chemical is not known to cause and cannot be reasonably anticipated to cause chronic health effects in humans. Third, the Petitioner must show that the chemical is not known to cause and cannot be reasonably anticipated to cause significant adverse effects on the environment because of its toxicity and its persistence or tendency to bioaccumulate in the environment.

In 1999 EPA found that BP had satisfied the first of these requirements completely for acetonitrile, that BP had ruled out all chronic health effects with the exception of neurotoxicity and mortality, and that BP had satisfied the third category of requirements with the single exception of not demonstrating that acetonitrile will not contribute significantly to the formation of ozone in the ambient air, which EPA concluded would be a ground for retaining acetonitrile on the TRI list. In short, EPA concluded that acetonitrile should be retained on the list because of concerns about: (1) chronic neurotoxicity, (2) chronic mortality effects, and (3) potential contribution to ozone formation as a volatile organic compound ("VOC").

Since EPA's initial denial of the BP Petition for acetonitrile, BP submitted additional information to EPA on the chronic neurotoxicity issue that caused EPA to reverse its position and conclude in December 2000 that acetonitrile is not a chronic neurotoxicant.

1/ Because EPCRA was passed as Title III of the Superfund Amendments and Reauthorization Act of 1986 ("SARA"), the statute is also commonly known as "SARA Title III."

This Petition summarizes the grounds for delisting that were presented in the previous submissions by BP and provides additional information on the remaining two issues sufficient to reverse EPA's conclusions about chronic mortality effects and the potential contribution of acetonitrile to ozone formation. Specifically, BP demonstrates that under generally accepted scientific principles, which the statute directs EPA to follow, chronic mortality is not an issue for concern, because the only exposures that might occur in communities would be at levels that EPA has already determined under its Integrated Risk Information System ("IRIS") will be safe for lifetime exposures to acetonitrile. EPA cannot make a decision to retain acetonitrile on the TRI list of toxic chemicals that contradicts extremely conservative risk assessment and risk management decisions reached under IRIS.

Finally, with respect to the concerns that EPA has expressed about acetonitrile's potential contribution to ozone formation as a VOC, BP demonstrates that EPA's Office of Air Quality Planning and Standards ("OAQPS") has already concluded that acetonitrile does not have sufficient photochemical reactivity to contribute to ozone formation that would pose any significant health risks. The only thing standing in the way of a favorable decision on this issue is a failure to date of EPA's TRI Program and OAQPS to reach a coordinated decision on the issues of the photochemical reactivity and the potential environmental toxicity of acetonitrile. In this Petition, BP specifically asks these two EPA offices to make a coordinated decision on these issues and to rule in accordance with the evidence that shows that acetonitrile should be deleted from the list of toxic chemicals under TRI and from the definition of VOCs under the Clean Air Act. As a result, this Petition provides the necessary support for EPA to eliminate the last two concerns that stand in the way of EPA's determination that acetonitrile should be deleted from the TRI list of toxic chemicals maintained under Section 313 of EPCRA.

PETITION OF BP CHEMICALS INC. TO DELETE ACETONITRILE FROM THE TRI LIST OF TOXIC CHEMICALS

TABLE OF CONTENTS

LIST OF TABLES

LIST OF EXHIBITS

P. U. S. EPA, Toxicological Review of Acetonitrile at 8-9 (CAS No. 75-05-8) (January 1999).

Q. Letter from Dr. Alan M. Hoberman to Dale E. Strother (March 26, 2002).

R. EPA, Hazard Assessment for Acetonitrile 4 (June 8, 1998).

S. *Fertilizer Institute Inc. v. Browner*, Civ. Action No. 98-1067, Slip op., (D.D.C., April 15, 1999).

T. Memorandum to Mario Doa, Toxics Release Inventory Branch, EAD, from G. Tom Helms, Ozone Policy and Strategies Group, AQSSD, "Photochemical Reactivity of Acetonitrile" (Oct. 1, 1998).

PETITION OF BP CHEMICALS INC. TO DELETE ACETONITRILE FROM THE TRI LIST OF TOXIC CHEMICALS

BP Chemicals Inc. ("BP") hereby petitions the United States Environmental Protection Agency ("EPA") to remove acetonitrile (Chemical Abstracts Service Number 75-05-8) from the list of Toxic Chemicals at 40 C.F.R. § 372.65 that are subject to the reporting requirements of the Toxic Release Inventory ("TRI"). This BP petition is filed under section 313(d)(3) of the Emergency Planning and Community Right-to-Know Act of 1986 ("EPCRA"). It builds upon a previous petition filed in 1998 by BP and the GNI Chemical Company ("GNICC") ("the 1998 Petition").[2/] That petition was denied by EPA on February 24, 1999, but only after determining that BP and GNICC had satisfied a number of the requirements for demonstrating that acetonitrile should be deleted from the TRI list of toxic chemicals.[3/] At the same time that EPA denied the 1998 Petition, EPA offered to provide an expedited review of any additional information, data, and studies relating to the end point of concern provided by BP to determine how this additional information might affect EPA's decision.[4/] BP Amoco accepted EPA's offer [5/] and provided additional information in a series of submissions and presentations. This petition builds upon the 1998 Petition, EPA's 1999 Decision on that petition,[6/] and subsequent BP demonstrations and EPA determinations on the status of acetonitrile under the EPCRA section 313 delisting criteria.

This petition demonstrates that acetonitrile meets all of the section 313 criteria for delisting. First, acetonitrile is not known to cause and cannot be reasonably anticipated to cause significant adverse human health effects at concentrations that are reasonably likely to exist beyond facility boundaries as a result of continuous or frequently recurring releases. EPA determined this to be true when it acted on the previous 1998 Petition. There is no new evidence since 1999 that provides any basis for changing that assessment, and the information on releases shows an overall reduction rather than increase in annual release levels.

2/ Petition of BP Chemicals, Inc. and the GNI Chemical Company to Delist Acetonitrile from the List of Toxic Chemicals Under the Toxic Release Inventory (February 3, 1998) (Exhibit A).

3/ Acetonitrile; Community Right-to-Know Toxic Chemical Release Reporting, 64 Fed. Reg. 10597 (March 5, 1999) ("the 1999 Decision") (Exhibit B).

4/ Letter to S. Patrick Presley, BP Amoco, from Dr. William H. Sanders III, EPA Director of OPPT (Feb. 24, 1999) (Exhibit C).

5/ Letter to Dr. William H. Sanders III, EPA Director of OPPT, from S. Patrick Presley, BP Amoco (March 5, 1999) (Exhibit D).

6/ Because this Petition builds upon the 1998 Petition, the administrative record for the 1998 Petition is hereby incorporated by references. EPA is requested to include that administrative record as a part of the record for this Petition.

Second, acetonitrile is not known to cause and cannot be reasonably anticipated to cause cancer or teratogenic effects or serious irreversible reproductive dysfunction, neurological disorders, heritable genetic mutations, or other chronic health effects. Although EPA has stopped short of making this determination to date, the evidence in this petition provides the basis for such a determination.

Third, acetonitrile is not known to cause or reasonably likely to cause significant adverse effects to the environment because it is not toxic or persistent and does not readily bioaccumulate. EPA determined this to be true in its 1999 Decision, and there is no evidence to contradict EPA's prior determination on this point. Because acetonitrile meets all three of these criteria, it should be removed from the list of Toxic Chemicals at 40 C.F.R. § 372.65.

I. INTRODUCTION

A. Description of Acetonitrile

Acetonitrile, with chemical formula CH_3CN, is a volatile, colorless liquid with a sweet, ether-like odor. Its synonyms include cyanomethane, ethanenitrile, nitrile of acetic acid, methyl cyanide, ethyl nitrile, and methanecarbonitrile. Table 1 presents selected properties of acetonitrile.

Table 1. Physical Properties of Acetonitrile

Properties	Value	Reference[7/]
Relative molecular mass	41.05	---
Appearance	colorless liquid	Budavari (1989)
Odor	ether-like	Budavari (1989)
Boiling point	81.6 °C (760 mmHg)	Budavari (1989)
Freezing point	-45.7 °C -44 to -41 °C	Grayson (1985) Verschueren (1983)
Specific gravity	0.78745 (15/4 °C) 0.7138 (30/4 °C)	Grayson (1985) Grayson (1985)

7/ All references are cited in: International Programme on Chemical Safety, Acetonitrile, Environmental Health Criteria 154, World Health Organization, Geneva (1993) ("WHO Monograph").

Properties	Value	Reference[7/]
Vapor density	1.42 (air = 1)	Clayton and Clayton (1982)
Refractive index (N_D)	1.34604 (15 °C)	Clayton and Clayton (1982)
	1.33934 (30 °C)	Clayton and Clayton (1982)
Solubility in water	infinitely soluble	Clayton and Clayton (1982)
Vapor pressure		
at (15.5 °C)	7.32 kPa (54.9 mmHg)	U.S. EPA (1984)
at (20.0 °C)	(74.0 mmHg)	Verschueren (1983)
at (30.0 °C)	(115.0 mmHg)	Verschueren (1983)
Water azeotrope	boiling point 76 °C water content 16%	U.S. EPA (1984)
Log P (octanol/water partition coefficient)	-0.38	Leo, et al. (1971)
	-0.34	Verschueren (1983)
Flash point	5.6 °C (open cup)	Reynolds (1982)
	12.8 °C (closed cup)	Reynolds (1982)
Autoignition temperature	524 °C	Sax and Lewis (1989)
Explosive limits in air (% by volume)	lower 4.4	Grayson (1985)
	3.05	Prager (1985)
	upper 16.0	Grayson (1985)
	17.0	Prager (1985)

Acetonitrile is infinitely soluble in water and readily miscible with ethanol, ether, acetone, chloroform, carbon tetrachloride, and ethylene chloride; it is immiscible with many saturated hydrocarbons. Although one of the more stable nitriles, acetonitrile undergoes typical nitrile reactions and is used to produce many types of nitrogen-containing compounds, such as amides, amines, higher molecular weight mono- and dinitriles, halogenated nitriles, ketones, isocyanates, and heterocycles (pyridines and imidazolines). The chemical can be timerized to S-trimethyltriazine and has been telomerized with ethylenes and copolymerized with alpha-

epoxides. Acetonitrile produces hydrogen cyanide when heated to decomposition or when reacted with acids or oxidizing agents.[8/]

Acetonitrile is obtained as a coproduct of acrylonitrile production by high temperature catalytic reaction between propylene, ammonia, and air. The crude acrylonitrile contains between three and ten percent acetonitrile, and is obtained by fractional distillation after cooling. Specifications for commercial acetonitrile are provided in Table 2.

Table 2. Specification for Commercial Acetonitrile[9/]

Specification	Value
Specific gravity (at 20 °C)	0.783-0.787
Distillation range, °C	
initial min.	80.5
end pt., max.	82.5
Purity (min.), wt %	99.0
Acidity (max.), wt %	0.05
Copper (max.), ppm	0.5
Iron (max.), ppm	0.5
Water (max.), wt %	0.3
Color (max.), Pt-Co	15

Because of its excellent solvent properties and relatively low boiling point, acetonitrile is used as a starting material and recoverable reaction medium for the synthesis of many chemicals, pharmaceuticals,[10/] pesticides,[11/] and in the manufacture of photographic color film. The chemical is used as a solvent in extraction procedures, such as butadiene extraction from C4 streams and isoprene from C5 streams, dissolution of cationic textile dyes, recrystallization of steroids, extraction of fatty acids from animal and vegetable oils, removal of tars, phenols, and coloring matter from petroleum hydrocarbons, and solvent for spinning fibers

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8/ Encyclopedia of Chemical Technology (1981), Kirk Othmer, ed., 3d ed., vol 15, at 895-897 ("Kirk Othmer").

9/ Kirk Othmer, at 896 (Table 6).

10/ Examples of pharmaceuticals in which acetonitrile is used as a reaction/separation solvent include antibiotics, HIV drugs, an anti-viral drug, drugs for diabetes, an anti-cholesterol drug, an anti-hypertension drug, an anti-depressant, as well as other drugs used for staph infections, schizophrenia, and as anti-bacterials.

11/ Acetonitrile is used as a raw material for insecticides, such as in the growing of cotton, vegetables, and turf at golf courses.

and casting and molding of plastics.[12/] Acetonitrile is also used widely in research and analytical laboratories as a solvent for genetic engineering research and in high performance liquid chromatography ("HPLC"), based on its consistent purity and its low ultraviolet light cutoff. The compound is also used as an inert medium in physico-chemical investigations and as a solvent in non-aqueous titrations.[13/]

B. Criteria for Deleting a Chemical from the List of Toxic Chemicals

Section 313(e)(1) of EPCRA authorizes any person to petition the EPA to delete a chemical from the list of Toxic Chemicals at 40 C.F.R. § 372.65.[14/] A delisting petition will be successful if EPA finds that the Toxic Chemical does not meet any of the criteria at section 313(d)(2) of EPCRA for placing a chemical on the list.[15/] Section 313(d)(2) contains three sets of criteria:

(A) That the chemical:

- (i) is known to cause or can be reasonably anticipated to cause significant adverse acute human health effects
- (ii) at concentrations that are reasonably likely to exist beyond facility site boundaries
- (iii) as a result of continuous, or frequently recurring, releases;[16/]

(B) That the chemical is known to cause or can be reasonably anticipated to cause in humans:

- (i) cancer or teratogenic effects, or
- (ii) serious or irreversible:
  - (I) reproductive dysfunction,
  - (II) neurological disorders,
  - (III) heritable genetic mutations, or
  - (IV) other chronic health effects;[17/] and

12/ *See also* J. Miller, Economic Analysis of the Proposed Deletion of Acetonitrile from the EPCRA Section 313 List of Toxic Chemicals (March 30, 1998) (Exhibit E).

13/ Kirk Othmer, at 897.

14/ 42 U.S.C.A. § 11023(e)(1).

15/ 42 U.S.C.A. §11023(d)(3).

16/ 42 U.S.C.A. §11023(d)(2)(A).

17/ 42 U.S.C.A. §11023(d)(2)(B).

(C) That the chemical is known to cause or can be reasonably anticipated to cause a significant adverse effect on the environment because of its:

(i) toxicity,
(ii) toxicity and persistence in the environment, or
(iii) toxicity and tendency to bioaccumulate in the environment. [18/]

EPCRA section 313 states further that its "determination under this paragraph shall be based on generally accepted scientific principles or laboratory tests, or appropriately designed and conducted epidemiological or other population studies, available to the Administrator."[19/] This means that EPA must rely on fundamentally sound applications of scientific principles as the basis for its interpretations of the underlying evidence. This especially includes EPA's own scientific determinations.

While the criteria for chronic toxicity do not directly address exposure, as do the criteria for acute toxicity, the courts have upheld EPA's conclusion that EPCRA grants the Agency the discretion to consider exposure information in making a determination based on chronic toxicity, and have recognized the Agency's policy of considering exposure in cases of chemicals with low to moderately low toxicity.[20/]

The Agency has drafted interpretive guidelines, the Hazard Assessment Guidelines for Listing Chemicals on the Toxic Release Inventory[21/] ("TRI Guidelines") for reviewing petitions to add or delete chemicals from the list of Toxic Chemicals. The TRI Guidelines divide chemicals into three groups based on chronic and environmental toxicity: high, medium, and low priority for listing.[22/] Chemicals in the middle category are to be considered on a case-by-case basis by EPA after a hazard evaluation.[23/] During the discussion of the appropriate criteria, this petition will categorize chemicals according to the TRI Guidelines, as revised during the "megalisting" rulemaking.

This petition will examine the human and environmental toxicity of acetonitrile under each of the three statutory criteria in section 313(d)(2) of EPCRA. Where EPA has

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18/ 42 U.S.C.A. §11023(d)(2)(C).

19/ EPCRA §313(d)(2), 42 U.S.C.A. §1023(d)(2).

20/ *Troy Corporation v. Browner*, 120 F.3d 277, 286 (D.C. Cir. 1997).

21/ U.S. Environmental Protection Agency, Hazard Assessment Guidelines for Listing Chemicals on the Toxic Release Inventory, revised draft (May 26, 1992) ("TRI Guidelines") (Exhibit F).

22/ TRI Guidelines, at 6. The TRI Guidelines used the terms "sufficient for listing," "may be sufficient for listing," and "insufficient for listing." *Id.* These terms were supplanted in EPA's so-called "megalisting" proposal without any change in the subjective content of the categories or the procedure to assess chemicals in those categories. 59 Fed. Reg. 1788, 1790 (1994).

23/ TRI Guidelines, at 4.

already made determinations in its 1999 Decision or in subsequent decisions based on additional information submitted by BP, those decisions will be summarized by reference to the evidence on which they were based. The evidence supporting the decisions and the decision documents are included as exhibits to this Petition. BP believes that EPA has already made all of the necessary scientific and technical decisions that support delisting for acetonitrile and that, therefore, EPA should grant this Petition and delist acetonitrile.

II. EPA Has Already Determined that Acetonitrile Satisfies the Agency's Delisting Criteria for Acute Toxicity.

The 1998 Petition demonstrated that acetonitrile is not found at environmental levels that could cause acute health effects, and thus does not meet the statutory criteria for adverse acute health effects to support inclusion on the list of Toxic Chemicals. The petition's discussion of the human clinical effects examined both experimental studies and poisoning cases, and described the clinical effects of acute exposure to acetonitrile. Only one study experimentally examined the effects of acetonitrile intake, and found mild transitory effects at 80 and 160 ppm. Several cases of accidental or intentional poisoning have been reported. While exposure was high, precise concentration data are lacking. Two cases involved inhalation. In one case, thirteen workers were exposed to paint containing 30-40% acetonitrile and paint thinner containing 95% acetonitrile. Although one fatality occurred, all other exposed individuals recovered fully and exhibited no lasting effects.

The clinical effects of exposure to acetonitrile are driven by the metabolism of the chemical into cyanide and thiocyanate, which are then excreted via the urine. The transformation of acetonitrile to cyanide occurs at a slower rate than for other nitriles. Most toxic effects are apparently due to cyanide production. Only huge intakes of acetonitrile result in death. More common exposures can result in a variety of sublethal respiratory, neurological, and other effects.

Data from laboratory animals on adverse effects of acute exposure were examined in detail in the 1998 Petition, including exposure concentration (*i.e.*, dose) data. The Petition examined inhalation data first because it is the primary potential route of exposure for acetonitrile. But the Petition also considered other routes of exposure, such as gavage, intraperitoneal, and intravenous. In the final analysis, acetonitrile has been shown to have a low order of acute toxicity following administration to experimental animals. When compared to other aliphatic mononitriles in the homologous series, acetonitrile has been shown to be far less toxic by several acute exposure routes.

A. Short-Term Exposure to Acetonitrile Will Not Occur at Concentrations Able to Cause Adverse Effects.

To be listed or retained on the list of Toxic Chemicals, EPCRA requires a showing that the chemical satisfies all of the criteria for acute toxicity, *i.e.*, that each of the following criteria is satisfied: (1) the chemical is known to cause or can be reasonably

anticipated to cause significant adverse acute human health effects, (2) the adverse effects occur at concentrations that are reasonably likely to exist beyond facility site boundaries, and (3) those concentrations are a result of continuous, or frequently recurring, releases.[24/] While acetonitrile does satisfy the first criterion, the 1998 Petition demonstrated and EPA concluded that the second and third criteria — and particularly the second criterion — are not satisfied.

The 1998 Petition used air modeling to assess exposure because the releases that could result in human exposure were air emissions. The 1998 Petition used the TRI data for 1995, the most recent year for which data were available at that time. Of the 28,866,549 pounds of acetonitrile reported as released into the environment for 1995,[25/] 27,837,181 pounds were injected underground, and not available for exposure to the human population.[26/] Of the remaining releases (nonpoint and stack air emissions, surface water discharges, and releases to land), air emissions constituted 99.3% of the releases.[27/]

For the 1998 Petition, BP and GNICC commissioned Trinity Consultants ("Trinity") to model maximum likely long-term air emissions from facilities that manufactured or processed acetonitrile. Ambient levels of acetonitrile were modeled using the ISCLT3 model, following the guidance in EPA's TRIAIR Users Guide. Trinity used data from the ten facilities with the largest total air emissions of acetonitrile in 1995, as reported to the TRI. The results of the modeling study showed that, at the fenceline of each of the ten facilities,[28/] acetonitrile is found at very low levels. Maximum likely annual concentrations at the fenceline ranged from 2.844 to 38.240 µg/m3. Based on this analysis, the 1998 Petition concluded that acetonitrile does not meet the second criterion for acute toxicity—the occurrence of adverse effects at concentrations that are reasonably likely to exist beyond site boundaries—and thus does satisfy the delisting criteria for acute toxicity.

24/ 42 U.S.C.A. §11023(d)(2)(A).

25/ U.S. EPA, 1995 Toxics Release Inventory, Public Data Release, EPA 745-R-97-005, at 74 (April 1997) ("TRI Report").

26/ *See*, Robert Van Voorhees, *Removed from the Environment,* 18 Env. Forum 23 (2001) (Exhibit G).

BP continues to urge the Agency to not classify underground injection as a "release" on the TRI, and to report Class I injection instead as a waste management method that results in "contained disposal." BP supports the Supplemental Petition filed by the Underground Injection Task Group of the American Chemistry Council on October 16, 2001.

27/ Nonpoint air emissions were 698,612 pounds, stack emissions 323,370 pounds, surface water discharges 7,474 pounds, and releases to land 12 pounds. TRI Report at 74.

28/ Trinity Consultants, Refined Modeling for Ten Acetonitrile Emitting Facilities, prepared for BP Chemical and GNICC Group, Inc., Project 974401.0200 (January 1998) (Exhibit H).

B. EPA Found Inadequate Evidence to Support Listing for Acute Effects.

EPA observed in its decision on the 1998 Petition that "[t]he only available data regarding acute effects of acetonitrile in humans are from reports of accidental poisonings resulting from acute exposures." But, as EPA also noted, "It is likely that these acute exposures were at concentrations in excess of 500 ppm." EPA also concluded that other effects might occur in animals at 500 ppm.[29] Accordingly, EPA reviewed the exposure assessment conducted by BP and conducted an assessment of its own: "EPA performed exposure assessments to determine whether acute health effects from acetonitrile would occur at concentrations reasonably likely to exist beyond the facility site boundaries as a result of continuous, or frequently recurring, releases."[30/] EPA had General Sciences Corporation ("GSC") conduct modeling using TRI data for both 1995 and 1996.[31/] "Short-term (acute exposure) air concentrations were estimated using the SCREEN3 and ISCST3 models."[32/] Using the SCREEN3 model, EPA concluded that "the estimated air concentrations of acetonitrile beyond facility site boundaries at sites with fugitive air emissions greater than 10,000 kilograms per year (kg/year) for 1995 and 1996 ranged from 4 to 36 milligrams per cubic meter (mg/m^3) (2.4 to 22 ppm) for 1 hour, and 1 to 14 mg/m^3 (0.9 to 8 ppm) for 24 hours, respectively."[33/] GSC also used the ISCST3 model and applied it to TRI data from both 1995 and 1996:

> Based on the 1995 data and the ISCST3 model, the 1 and 24 hours short-term (acute exposure) acetonitrile concentrations in air, at 100 meters distance from the source center of highest release, in the direction of highest concentration, are 16 and 2.3 mg/m^3 (or 9.52 and 1.37 ppm), respectively. Under the same model scenario, the 1996 data gave an estimated 23 and 3.3 mg/m^3 (or 13.5 and 2.0 ppm) of acetonitrile concentrations in air for the 1 and 24 hour short-term exposure, respectively.[34/]

With all of these exposure assessment results in hand, EPA concluded that "[t]hese estimated values of acetonitrile in air are well below those concentration levels that

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29/ 64 Fed. Reg. at 10,599.

30/ 64 Fed. Reg. at 10,601.

31/ Annett Nold, Exposure Assessment for Acetonitrile (CAS Number 75-05-8) in Response to Delisting Petition (July 10, 1998) (Exhibit I); General Sciences Corporation, Modeling Support for Exposure Assessment of Acetonitrile (July 10, 1998) (Exhibit J).

32/ 64 Fed. Reg. at 10,601.

33/ 64 Fed. Reg. at 10,601.

34/ 64 Fed. Reg. at 10,601.

produced acute effects in animal studies."[35/] EPA also conducted an exposure assessment for drinking water and concluded that there was no potential for exposure to lead to adverse effects. The overall conclusion stated by EPA was:

> Under the conditions modeled here EPA believes it is unlikely that concentrations of acetonitrile sufficient to cause acute toxicity will exist beyond a facility's boundaries as a result of continuous, or frequently reoccurring, releases. This is because the exposure concentrations that resulted from the modeling (9.52 and 1.37 ppm) are below the concentrations that have caused acute toxicity in laboratory animals (500 ppm).[36/]

C. Conclusions on the Absence of an Acute Toxicity Concern Remain Valid.

The conclusions demonstrated in the 1998 Petition and confirmed by EPA in its 1999 Decision remain valid today, because there have been no new studies that have changed the basis for the scientific conclusions about the concentration levels at which acute toxic effects may potentially occur. In addition, a comparison of the TRI data from subsequent years shows that the conclusions reached on the basis of 1995 and 1996 data will remain valid. As shown in Table 3, there has been no significant increase in the TRI release numbers for the 10 facilities with the largest air release numbers since 1996. Accordingly, it remains true that concentrations of acetonitrile sufficient to cause acute toxicity will not exist beyond a facility's boundaries as a result of continuous, or frequently recurring, releases.

III. EPA Has Already Concluded that Acetonitrile Satisfies the Agency's Criteria for Delisting Based on Chronic Toxicity for All Effects Other than Mortality.

A. In 1999 EPA Eliminated Concerns Over Chronic Effects Other than Neurotoxicity and Mortality.

The 1998 Petition concluded that acetonitrile also satisfied the delisting criteria for chronic toxicity effects, but EPA did not accept this conclusion for all chronic effects. EPA concluded instead that "[t]here is sufficient evidence to support a high level of concern for potential neurotoxicity and death following repeated exposure to acetonitrile."[37/] At the same time, however, EPA agreed to reconsider this conclusion based on any new evidence that BP could provide. Following additional review, EPA ultimately agreed that there was insufficient evidence to support any continuing concern over neurotoxicity.[38/] Consequently, EPA has

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35/ 64 Fed. Reg. at 10,602.

36/ 64 Fed. Reg. at 10,602.

37/ 64 Fed. Reg. at 10,602.

38/ Letter from Elaine Stanley, Director of EPA's Office of Information Analysis and Access, to Dale E. Strother of BP Amoco Chemicals, Inc. (December 22, 2000) (Exhibit K).

agreed that BP has demonstrated the absence of evidence to support a finding of chronic toxicity concerns on any basis other than mortality.

For each of the specifically enumerated chronic effects in section 313(d)(2)(B), EPA has concurred that there is no demonstrable basis for concern. For cancer, EPA concluded that "[b]ased on the results of the NTP studies, there is insufficient evidence to conclude that acetonitrile may or has the potential to cause cancer in humans."[39/] For mutagenicity, EPA concluded that "there is no basis for concern for potential heritable gene or chromosomal mutagenicity of acetonitrile."[40/] EPA also did not find evidence sufficient to support concerns for developmental or reproductive toxicity for acetonitrile.[41/] For other chronic effects, EPA found no specific evidence to support any particular concern over other chronic effects apart from mortality.

B. In 2000 EPA Agreed that the Scientific Evidence Does Not Support Concern Over Chronic Neurotoxicity for Acetonitrile.

As a basis for denying the 1998 Petition, EPA concluded that acetonitrile can reasonably be anticipated to cause serious or irreversible chronic neurotoxicity effects in humans at the relatively low dose of approximately 30 mg/kg/day. Based on this conclusion, EPA considered acetonitrile to have moderately high to high chronic neurotoxicity. In response to this determination, BP submitted additional information on neurotoxicity in a letter to EPA dated March 5, 1999 (Exhibit D), at a meeting and presentation on June 22, 1999, and in a subsequent submission on October 12, 1999 (Exhibit L). The additional information provided by BP specifically addressed the studies that EPA had cited as the basis for its conclusion about neurotoxicity. The additional information included, in particular, pathology reviews prepared by Dr. Robert Garman. Based on these submissions, BP concluded that "there is compelling evidence that acetonitrile is not a chronic neurotoxin."[42/]

EPA's initial conclusions were based, directly or indirectly, on four studies: Pozzani, et al.,[43/] Argus Research Laboratories ("Argus"),[44/] Du Pont,[45/] and NTP.[46/] The new

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39/ 64 Fed. Reg. 10,600.

40/ 64 Fed. Reg. 10,600.

41/ 64 Fed. Reg. 10,600.

42/ Letter to Dr. Jennifer Seed of EPA from Dale E. Strother of BP Chemicals (July 27, 2000) (Exhibit M).

43/ Pozzani, U.C., C.P. Carpenter, P.E. Palm, C.W. Weil, and J.H. Nair, An investigation of the mammalian toxicity of acetonitrile, J. Occup. Med. 12: 634-642 (1959) ("Pozzani study").

44/ Argus Research Laboratories, Inc., Embrya-fetal toxicity and teratogenicity study of acetonitrile in New Zealand White Rabbits (Segment II Evaluation), Project No. 419-001, Final Report, EPA Document No. 40-8446070, Fiche No. OTS0507279 (1984) ("Argus study").

information provided by BP on the first three studies – both individually and in combination – showed that these studies, when fully understood in light of this information, did not support concerns for neurotoxicity from chronic exposures. BP reasoned further that, when the only study of sufficient quality to meet generally accepted scientific standards ("the NTP study") is used to decide whether acetonitrile causes chronic or subchronic neurotoxic effects, it becomes clear that acetonitrile does not cause chronic or subchronic neurotoxic effects or mortality.

The Pozzani study was cited by the Agency for providing evidence of nonhuman neurotoxic effects. EPA's 1999 Decision listed brain hemorrhages, hyperexcitability, and overextension reflexes in rats, monkeys, and dogs as contributing to this conclusion.[47/] The 350 ppm level for onset of neurotoxic effects cited in the notice of denial also came from the Pozzani study.[48/] A June 22, 1999, letter from EPA[49/] acknowledged "some of the deficiencies" of the Pozzani study, and stated that the Agency did not rely solely on that study. The new information presented by BP showed the deficiencies in this study to be serious enough to discount it entirely, and to nullify any contribution that this study might make to the "several lines of evidence" of chronic neurotoxicity cited by EPA. These deficiencies involved both the observations of brain hemorrhages and behavioral effects and fundamental flaws in the experimental design – sample size, quality of test animals and chemicals, documentation of exposure levels – when judged against today's standards.

EPA's 1999 Decision also cited the Argus Laboratories Study as supporting a finding of chronic or subchronic neurotoxicity.[50/] As with the Pozzani study, the Argus study provides no reliable evidence of chronic or subchronic neurotoxicity. There are a number of factors that limit the conclusions that can be drawn from the Argus study. First, of twenty-five high-dose dams subject to the study, only two individuals exhibited any neurological signs. One individual that died exhibited neurological signs on the day before death, and a surviving dam

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45/ E.I. du Pont de Nemours and Company, Acute inhalation toxicity in rats with cover letter, Haskell Laboratory for Toxicology and Industrial Medicine, EPA Document No. 878220234, Fiche No. OTS0215023 (1968) ("Du Pont study").

46/ National Cancer Institute/National Toxicology Program, Toxicology and Carcinogenesis of Acetonitrile (CAS No. 75-05-8) in F344/N Rats and B6C3F1 Mice (inhalation studies), Carcinogenesis Technical Report Series; National Cancer Institute/National Toxicology Program; U.S. Department of Health and Human Services (1996) ("NTP study").

47/ 64 Fed. Reg. at 10,600.

48/ 64 Fed. Reg. at 10,600.

49/ Letter to S. Patrick Presley, BP America Inc., from Dr. William H. Sanders III, EPA Director of OPPT (June 22, 1999) (Exhibit N).

50/ 64 Fed. Reg. at 10,600.

exhibited signs on only one day prior to aborting her litter.[51/] Second, based on the day of onset of neurological signs in these two dams, the study is really an acute exposure study.

The limitations of the Argus Study for supporting any conclusions about chronic effects have been highlighted by Dr. Alan Hoberman, one of the study managers, who noted that "EPA has typically considered the exposure period used in a developmental toxicity study to be an acute exposure whenever a reference dose is calculated."[52/] Specifically, Dr. Hoberman attributes the five deaths observed in the study to acute effects:

> Five does died prior to scheduled sacrifice in the 30 mg/kg/day dosage group. The pattern of these deaths (days 12, 15, 17, 18, and 19) indicates that these deaths were caused by acute effects of the test substance.

In responding to a question about whether the findings of the Argus Study could support a conclusion of chronic toxicity, Dr. Hoberman states: "None of the findings in the full developmental toxicity study or in the dosage range can be separated from the acute toxicity of the test substance."[53/] Dr. Hoberman further notes that "the use of a pregnant rabbit at dosages designed to produce acute maternal toxicity would never be appropriate as a model for evaluating chronic toxicity."[54/] In his view, the only appropriate use that could be made of the Argus rabbit study for considering chronic effects would be "to provide information that would aid in dosage level selection for chronic studies in rabbits."[55/] Accordingly, the Argus rabbit study would not provide proper supporting evidence of chronic neurotoxicity even if the results were less equivocal than the two observations reported.

The Agency also cited the Du Pont study as supportive of a conclusion that "[o]ther laboratory studies also show that inhalation exposure to acetonitrile can adversely affect the nervous system of animals."[56/] The study is discussed in Section IV.C.2.iv. of the denial of the petition, under chronic neurotoxic effects.[57/] Yet, the study is clearly an acute exposure study — as evidenced by its title and the four-hour exposure period — and cannot be used to support any conclusions that observations of neurological signs are chronic rather than acute effects of acetonitrile.

51/ Argus study, Table 8, at A-26 and A-28.

52/ Letter to Dr. Robert Kapp from Dr. Alan Hoberman (July 26, 1999) (Exhibit L, Tab E).

53/ Hoberman Letter at 2.

54/ Hoberman Letter at 2.

55/ 1999 Hoberman Letter at 3.

56/ 64 Fed. Reg. at 10,600.

57/ *See* 64 Fed. Reg. at 10,599-10,600.

On December 22, 2000, Elaine Stanley wrote to advise BP that "EPA has completed its review of the additional information relating to the chronic neurotoxicity endpoint provided by Dr. Robert Garman of Consultants in Veterinary Pathology, Inc. and agrees with BP Amoco's contention that, based on the available data, there is no evidence to support treatment-related signs of neurotoxicity following exposure to acetonitrile."[58/] Consequently, the neurotoxicity endpoint no longer provides any justification for retaining acetonitrile on the TRI list of toxic chemicals.

IV. EPA Has Already Determined that Acetonitrile Also Exhibits No Significant Adverse Effects on the Environment.

The third set of criteria for reviewing a delisting petition is to determine whether the Toxic Chemical is known to cause or can be reasonably anticipated to cause a significant adverse effect on the environment because of its (i) toxicity, (ii) toxicity and persistence in the environment, or (iii) toxicity and tendency to bioaccumulate in the environment. The 1998 Petition demonstrated that acetonitrile does not exhibit any significant adverse effects on the environment. In its 1999 Decision, EPA agreed apart from its expression of concern over acetonitrile as a volatile organic compound ("VOC"), which we consider as a separate matter in section VIII of this Petition. EPA said: "Acetonitrile is of low concern with respect to direct ecotoxicity based on measured data and Quantitative Structure Activity Relationship ("QSAR") analysis." EPA stated further that, "[b]ased on the limited number of laboratory studies conducted to date, the terrestrial toxicity of acetonitrile is low" and that "[n]o published experimental data are available for evaluating its bioaccumulation."[59/] Accordingly, there is no basis for retaining acetonitrile on the TRI list unless EPA can support the listing solely on the basis of its supposition that acetonitrile contributes significantly to the formation of ozone and that this consideration is sufficient to support a TRI listing.

V. Remaining EPA Concerns About Potential Chronic Mortality Effects Do Not Support Retention on the Toxic Chemical List.

When EPA concluded that acetonitrile is not a chronic neurotoxicant, the Agency asserted nonetheless that acetonitrile can reasonably be anticipated to cause death as a chronic health effect. Under EPCRA, this is a determination that must be based on generally accepted scientific principles or laboratory tests, or appropriately designed and conducted epidemiological or other population studies, available to the Administrator.

The brief references to mortality in EPA's 1999 Decision rationale focused on three studies: the Pozzani Study, the Argus Laboratories Study, and the National Toxicology

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58/ Letter from Elaine Stanley, Directory of EPA's Office of Information Analysis and Access, to Dale E. Strother of BP Amoco Chemicals, Inc. (December 22, 2000) (Exhibit K). *See also*, Memorandum from Dr. Katherine Anitole to Amy Neuman, "OPPT/RAD Decision on Neurotoxicity Endpoint for Acetonitrile" (September 8, 2000) (Exhibit O).

59/ 64 Fed. Reg. 10,601.

Program ("NTP") Study. Reliance on these studies was reiterated in the September 2000 Memorandum.[60/] Careful review of these studies, however, shows mortality to be an acute rather than chronic effect, especially when considered in conjunction with the evidence available from human exposures to acetonitrile.

A. The Pozzani Study

The Federal Register Notice stated that "death [was] observed at concentrations of acetonitrile at or near 350 ppm (approximately 30 mg/kg/day)."[61/] This level comes from the Pozzani study. Further review of the Pozzani study, however, has indicated that this conclusion about mortality was overstated. In fact, no deaths were attributed to the 350 ppm exposure level; mortality was only reported at higher levels presumed to be 660 ppm and 2510 ppm. For the highest exposure level, the death of the monkey occurred after only the second exposure, clearly an acute effect.

In addition, the Pozzani study does not conform to generally accepted scientific standards and cannot serve as the basis for a determination of chronic toxicity. There are a number of problems reflected in the experimental design of the Pozzani study. First, the study involved a smaller than acceptable number of test animals: three rhesus monkeys exposed to 350 ppm for seven hours per day for ninety-one days[62/] and a group of four rhesus monkeys exposed to 330 ppm (one individual) for ninety-one days, 660 ppm (two individuals) for 23 and 51 days, and 2,510 ppm (one individual) for two days.[63/] Second, the study lacked documentation on the source and general health of the monkeys and on the purity of the acetonitrile used to generate the exposure conditions. In addition, these same animals had been used several months earlier for intravenous studies of acetonitrile and sodium thiocyanate (another cyanide liberating chemical) and may have been used in other studies as well. Third, no control animals were used in the study of monkeys. Fourth, the Pozzani study lacks data to assure consistency of — and to document — the actual exposure concentrations of acetonitrile.[64/] The authors acknowledge that they had no accurate means of measuring the concentrations of acetonitrile, and had no means for ensuring that the concentrations remained constant. The concentrations were calculated rather than measured. Consequently, there can be no assurance that the observed effects were due to the concentrations stated in the article. This shortcoming, by itself, renders this study unusable by EPCRA's standards because the study does not measure up to generally accepted scientific principles.

60/ Memorandum from Dr. Katherine Anitole to Amy Neuman, "OPPT/RAD Decision on Neurotoxicity Endpoint for Acetonitrile" (September 8, 2000) (Exhibit O).

61/ 64 Fed Reg. at 10,602.

62/ Pozzani study at 638.

63/ Pozzani study at 638.

64/ Pozzani study at 638.

Considering the lack of concentration data, in combination with the other deficiencies in experimental design, the Pozzani study is seriously flawed and therefore unreliable. A ninety-day primate study is not easily dismissed, but this conclusion was confirmed by the Agency in connection with its recent development of a reference concentration ("RfC") for acetonitrile. The section on Chronic Health Hazard Assessments for Noncarcinogenic Effects in the Integrated Risk Information System ("IRIS") report took note of the Pozzani Study, but concluded that the "experiment was limited because of inadequate study protocol and results and the absence of a control group of monkeys."[65/] In light of these deficiencies noted in the IRIS report, the study was not relied upon in the development of the RfC for acetonitrile,[66/] and cannot be relied upon by EPA in making a decision under EPCRA section 313.[67/] The IRIS rejection of the study represents the application of generally accepted scientific principles for interpretation; EPA cannot reverse course now and rely on this study to support its decision on the TRI listing for acetonitrile.

B. The Argus Laboratories Study

EPA also identified the Argus Laboratories Study ("rabbits repeatedly exposed during gestation")[68/] as supporting a finding of a chronic mortality effect.[69/] Based on the design, the Argus Study is really an acute exposure study. As noted above (page 12), the limitations of the Argus Study for supporting any conclusions about chronic effects were reiterated by Dr. Alan Hoberman, one of the study managers in a 1999 letter.[70/] In a more recent letter dated March 26, 2002, Dr. Hoberman has clarified and expanded his comments on the Argus Rabbit Study.[71/] Dr. Hoberman indicates that "it is much more likely that these deaths resulted from acetonitrile-induced gastrointestinal distress alone or in combination with the acute systemic effects of acetonitrile." Dr. Hoberman explained that the dosage-range study was conducted "to find a maternally toxic dose that could be tolerated for the exposure period (13 days) so that a sufficient number of term fetuses are available for evaluation." He added that "[t]he dosage-range study clearly demonstrated the acute toxicity of acetonitrile in rabbits." His reason for concluding that the five rabbit deaths observed in the study were attributable to acute effects is stated as follows:

65/ U.S. EPA IRIS Substance File – Acetonitrile, CAS RN 75-05-8, at 8 of 16 (Mar. 3, 1999) ("IRIS paper") (Exhibit L, Tab D); U. S. EPA, Toxicological Review of Acetonitrile at 8-9 (CAS No. 75-05-8) (January 1999) ("IRIS Toxicological Review") (Exhibit P).

66/ *See* Section IV.A of this paper for a further discussion of the IRIS RfC development.

67/ *See Troy Corporation v. Browner*, 120 F.3d 277, 293 (D.C. Cir. 1997), where the Court found EPA reliance on a study not accepted by IRIS to be evidence of "arbitrary and capricious agency action."

68/ EPA 2000 Memorandum at 2.

69/ 64 Fed. Reg. at 10,600.

70/ Letter to Dr. Robert Kapp from Dr. Alan Hoberman (July 26, 1999) (Exhibit L, Tab E).

71/ Letter from Dr. Alan M. Hoberman to Dale E. Strother (March 26, 2002) (Exhibit Q).

> Rabbit survivability is well-known to be affected by substances that disrupt the gut flora. I consider the anorexia and other gastrointestinal symptoms observed in these animals to be a significant confounder to determining the contribution that systemic chemical toxicity played in these death.

As a result, Dr. Hoberman stated "I could not support the use of these study results to predict the chronic toxicity of acetonitrile following inhalation exposure." His position is based on a number of concerns, including:

- Well established differences in the absorption and disposition of chemicals following bolus gavage doses and airborne exposures.
- The potential confounding effect of gastrointestinal distress observed following gavage doses, as discussed above [in his letter].
- This study did not follow generally accepted scientific design standards for either inhalation studies or chronic effect studies.

Accordingly, the Argus rabbit study would not provide proper supporting evidence of chronic effects even if the results were less equivocal than the two observations reported. Reliance on this study to draw conclusions about chronic effects does not comply with the EPCRA section 313(d) requirement to rely on "generally accepted scientific principles or laboratory tests" or "appropriately designed" studies.

C. The NTP Study

In its June 22, 1999, letter to BP Amoco, the Agency cited the NTP study, among others, to support the statement that acetonitrile causes delayed onset of adverse health effects, and hence adverse effects from chronic or subchronic exposure.[72/] A careful review of the data in the NTP study, however, reveals that delayed onset of health effects is not indicated by the study. Mortality reported in the study was either the result of acute exposures or could not be linked to exposure to acetonitrile.

1. Mortality in the Subchronic Study Was an Acute Effect.

The data on both rats and mice show that mortality is not a chronic effect of exposure to acetonitrile. Under generally accepted scientific principles, chronic is defined to be greater than 90 days exposure, but the two NTP subchronic studies lasted only thirteen weeks (91

72/ EPA's June 22 letter at 3.

days). In the thirteen-week mouse study, the mice that died during exposure (26 mice), with only three exceptions, died early in the study (*i.e.*, by the end of week 3). At the 1600 ppm dose level, all mice died by the third week of exposure. At 800 ppm, two females died during the second or third week of exposure; and two females and one male died during the sixth to thirteenth weeks. At 400 ppm, one female died during the second week. In the thirteen-week rat study, all deaths occurred during the first four weeks of exposure. Four of ten males exposed to 1600 ppm of acetonitrile died during the first week of exposure, and two more died by the fourth week. Three of the ten females that were exposed to 1600 ppm died during the first or second week of exposure; the rest survived to the end of the study. The only remaining rat (male) to die of exposure succumbed during the first week to levels of 800 ppm. With only three exceptions, all of the mortality in the subchronic exposure study occurred early during exposure, thus the mortality is properly described as the result of less than chronic exposure to acetonitrile. In light of the evidence, it seems most likely that the animals that died later in the study suffered acute effects from earlier exposure. Thus, even the later deaths are more likely to represent acute rather than chronic effects.

2. Mortality in the Chronic Study Was Unrelated to Exposure.

While deaths occurred in the chronic portion of the study, those deaths were not attributable to exposure to acetonitrile. In the two-year rat study using treatment groups of fifty-six animals, forty-four males in the control group died, and thirty-five males in the high-dose group died; thirty-one females in the control group died, and twenty-five females in the high-dose group died. In short, survival was better among the most highly exposed animals than among controls. For the exposed subjects, the study found "[t]wo-year survival . . . similar to . . . controls."[73/]

Similar results were found in the two-year mouse study; survival was actually ***better*** among the most highly exposed animals than among controls. Using treatment groups of sixty mice, twenty-eight individuals in the male control group died, and only seventeen died in the high-dose group. Thirty-two individuals in the female control group died, while twenty-eight died in the high-dose group. Again, exposed animals had a better survival rate than controls. The study found "[t]wo-year survival of exposed male and female mice was similar to that of controls, except that the survival of male mice in the 200 ppm group was ***significantly greater*** than that of the controls."[74/]

These results show that there were no statistically significant increases in mortality between the control and high-dose groups in either the rat or mouse studies. As a result, based on these two-year studies – the most scientifically sound and reliable studies available – no significant increase in mortality can be attributed to chronic exposure to acetonitrile.

73/ NTP Study at 6.

74/ NTP Study at 6 (emphasis added).

D. Other Evidence Also Shows Mortality Is an Acute Effect.

EPA observes that, for acetonitrile, "the main effects reported in humans are likely due to acute inhalation exposures to high concentrations."[75/] "Human data are limited to case reports of accidental poisonings in both workers and consumers of products containing ACN and one volunteer study."[76/] For the accidental poisonings, EPA concludes that "[i]t is likely that these acute exposures were at concentrations in excess of 500 ppm." For one of these incidents, it has been reported that sixteen painters were exposed to what could have been 10,000-50,000 ppm acetonitrile.[77] EPA reports that, "[a]t these high concentrations, acetonitrile affects the central nervous system producing excess salivation, nausea, vomiting, anxiety, confusion, hyperpnea, dyspnea, rapid pulse, unconsciousness, and convulsions, followed by death from respiratory failure."[78]

What is most noteworthy, however, is that EPA finds "based on the outcomes reported with accidental poisoning incidents, at sub-lethal concentrations these effects are reversible after removal from exposure."[79] The fifteen painters who did not die from the extremely high exposure did not experience any lasting effects from exposure. According to EPA, "[a]cute effects of acetonitrile in humans at concentrations less than 500 ppm consist of irritation of the mucous membranes."[80] No human deaths have been attributed to chronic exposure to acetonitrile. "No information was found on the adverse neurotoxic effects of long-term human exposure to acetonitrile."[81]

In the one voluntary study, humans breathed 40 ppm, 80 ppm and 160 ppm for four hours, with no subjective symptomatic response at any level except the 160 ppm level, where one of two subjects "experienced a slight transitory flushing of the face two hours after inhalation, and a slight feeling of bronchial tightness about five hours later. The latter symptom did not persist overnight."[82] "Both subjects stated they would have no hesitation about inhaling 160 ppm acetonitrile vapor again for a four-hour period."[83] In comparison, EPA has concluded

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75/ EPA, Hazard Assessment for Acetonitrile 4 (June 8, 1998) ("EPA Health Hazard Assessment") (Exhibit R).

76/ EPA Hazard Assessment at 6.

77/ Amdur, M.L., Accidental group exposure to acetonitrile, J. Occup. Med. 1(12): 627-633 (1959). *See also* Willhite (1981), citing NIOSH (1974) for the concentration levels.

78/ 64 Fed. Reg. at 10,599.

79/ EPA Hazard Assessment at 16.

80/ 64 Fed. Reg. at 10,599.

81/ 64 Fed. Reg. at 10,600.

82/ Pozzani study at 640-41.

83/ Pozzani study at 640-41.

that "[s]hort-term (24-hour) estimates of concentrations of ACN in the ambient air around industrial sites with the highest releases using the SCREEN3 model range from 1-10 mg/m^3 or ≤ 7 ppm."[84] These concentrations are far below any levels thought to create any acute effects or even symptoms in humans or animals.

Based on this overview of the evidence on the potential mortality effects of acetonitrile exposure, we are confident that the appropriate conclusion is that mortality is a potential acute rather than chronic effect. Moreover, based on EPA's own conclusions, mortality effects will not occur at any "concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases." EPCRA §313(d)(2)(A), 42 U.S.C. §11023(d)(2)(A). If EPA were to conclude that mortality is a chronic effect of acetonitrile exposure, it would be contrary to generally accepted scientific principles — and thus contrary to EPCRA — for EPA to fail to consider exposure levels. Mortality is a potential effect from exposure to almost any substance at high enough concentrations for long enough periods. EPA cannot justify listing or retaining a chemical simply because there may be a potential mortality effect. Accordingly, EPA should proceed to delete acetonitrile from the list of toxic chemicals in accordance with section 313(d)(3).

VI. Generally Accepted Scientific Principles Require EPA to Delist Acetonitrile.

EPA must delist acetonitrile unless the Agency determines that exposure of humans will occur at levels above the RfC that EPA has established for chronic inhalation toxicity. The listing/delisting provisions of EPCRA require, among other factors, that determinations be "based on generally accepted scientific principles." EPCRA § 313(d)(2), 42 U.S.C. § 11023(d)(2). Because EPA's National Center for Environmental Assessment ("NCEA") has established in IRIS an RfC for acetonitrile below which inhalation exposure is considered safe, generally accepted scientific principles require EPA to determine whether there is likely to be any exposure at concentration levels above that RfC level. If not, acetonitrile should be deleted from the list of toxic chemicals.

A. EPA has Established a Safe Level for Chronic Exposure to Acetonitrile.

IRIS is an electronic data base into which EPA has consolidated information on human health effects that may result from exposure to various chemicals in the environment. IRIS is maintained by NCEA within EPA's Office of Research Development. NCEA includes health assessment information in IRIS only after a comprehensive review of chronic toxicity data

84/ EPA Hazard Assessment at 4.

by EPA health scientists across the Agency.[85/] The health assessment information in IRIS is intended to represent a consensus reached in the review process. Past court decisions have noted this process, stating that under EPCRA, IRIS "is generally accepted as a reliable source of information on the potential hazardous effects of those chemicals that are included in IRIS."[86/]

On March 3, 1999, NCEA established an IRIS reference concentration for chronic inhalation exposure (RfC) for acetonitrile. EPA has explained that the RfC can be used to estimate a level of exposure at or below which no adverse effect is expected to occur. In the IRIS Summary for acetonitrile, EPA states: "In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."[87/] In other words, the RfC is a level that EPA has determined is a safe level for exposure even for the subgroups in the population that are expected to be the most sensitive to the effects of acetonitrile.

It must be noted here that BP does not find the conclusions of the IRIS analysis for acetonitrile to be entirely acceptable because those conclusions resulted in the establishment of an excessively conservative RfC, as will be explained below. But that is an issue to be addressed directly with the NCEA, and BP expressly reserves the right to seek an upward revision in the RfC for acetonitrile. For purposes of this petition, however, the significance of the RfC is simply underscored by its excessively stringent nature because the levels of acetonitrile existing beyond facility boundaries will not exceed even this low level. Thus, the use of IRIS findings by EPA for the purpose of delisting acetonitrile would be acceptable and is indeed compelled by generally accepted scientific principles. The RfC is an extremely conservative measure that will protect against any lifetime risks of mortality effects from chronic exposures to acetonitrile.

85/ IRIS Toxicological Review at 1 (1999) (Exhibit P):

> Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

http://www.epa.gov/iris/subst/0205.htm#reforal

86/ *National Oilseed Processors*, 924 F.Supp. 1193, 1200 (D.D.C. 1996).

87/ IRIS Toxicological Review at 1, www.epa.gov/iris/subst/0205.htm, (Exhibit P).

IRIS used the NTP subchronic studies and specifically the mortality endpoint to establish the RfC for acetonitrile.[88/] In short, EPA has determined that there is a safe lifetime exposure level for acetonitrile even considering the potential for chronic mortality effects.

B. The RfC for Acetonitrile Is Excessively Conservative.

BP does not dispute that the RfC for acetonitrile represents a safe level for human exposure, but BP maintains that the RfC is excessively conservative because it includes multiple and duplicative margins of safety. EPA used three different uncertainty factors and an additional modifying factor which produced an RfC for acetonitrile that is three orders of magnitude below the no observed adverse effect level (NOAEL).[89/] BP objects to the RfC for acetonitrile because it is unduly conservative for the following reasons.

First, EPA should have given more consideration to species differences between the mouse and humans and should have chosen the more appropriate surrogate. The mouse is very sensitive to aliphatic nitriles and cyanide toxicity. Physiology is important because the mouse inhales approximately 5-fold more air per unit of body weight than humans. Based on work with acetonitrile and other nitriles, human metabolism is more like the rat than the mouse. The IRIS assessment identified a NOAEL of 400 ppm for the rat as compared with a NOAEL of 200 ppm for the mouse. IRIS Toxicological Review at 7. Consequently, the starting point for the RfC is half of what it should have been.

Second, the RfC included a modifying adjustment for forestomach lesions in mice, but the relevance of forestomach lesions to humans is questionable. Humans do not have forestomachs, and rodent forestomachs are very sensitive to irritation. In addition, inhalation is unlikely to play a major role in the development of forestomach lesions, which are more likely the result of preening. "It is likely that preening activities and/or mucociliary clearance, resulting in oral ingestion of CAN, play a central role." IRIS Toxicological Review at 8. As noted by EPA, studies indicate that for rats, "60% of the pelt burden was calculated to be ingested following whole-body exposure." *Id.* Furthermore, as EPA noted, "[t]he absence of these lesions in the rat is puzzling." *Id.*

Third, EPA applied four separate adjustments to the NOAEL in deriving the RfC. A factor of 3 was used for interspecies extrapolation, a full factor of 10 was used to protect sensitive human subpopulations, and 3 was applied for database deficiencies (*e.g.*, reproductive endpoints, hematology in mice). Because two factors of 3 coalesce to a 10, a total uncertainty factor of 100 was used.

In addition, a modifying factor of 10 was applied because of the uncertain role that inhalation may have played in the development of the concentration-related increase in the

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88/ *Id.* Section I.B.2.

89/ The NOAEL is an experimentally determined dose at which there is no statistically or biologically significant indication of the toxic effect of concern.

incidence of forestomach lesions in both male and female mice. In light of the uncertainties about the potential contribution of inhalation, a separate adjustment for uncertainty due to forestomach lesions is very difficult to justify. The use of a full factor of 10 for this adjustment is even more difficult to rationalize because the lowest dose at which forestomach lesions were observed was 50 ppm, or 25% of the selected NOAEL. Thus, no modifying factor greater than 4 could possibly be justified."

Furthermore, the use of a modifying factor in addition to uncertainty factors has recently been questioned. EPA's Reference Dose Technical Panel has concluded that the purpose of the modifying factor is sufficiently subsumed in the uncertainty factors and is therefore recommending discontinuation of any use of a separate modifying factor. Specifically, the panel found that a modifying factor had only been used in conjunction with seven chemicals addressed in IRIS. The panel considered each of the cases, including acetonitrile, and concluded that the uncertainties intended to be addressed through the modifying factor could have been subsumed under other uncertainty factors. As a result, the RfC for acetonitrile should be an order of magnitude higher than ultimately established in the IRIS review process. The bottom line for purposes of this Petition is simply that the RfC established for acetonitrile is extremely conservative and, therefore, all the more appropriately considered to define a safe level for lifetime exposures to acetonitrile.

C. Although Overly Conservative, the RfC Sets a Safe Level for Exposure that EPA Must Consider in Making a Delisting Decision for Acetonitrile.

Through IRIS, the Agency has made a determination that there is a concentration level below which it is safe for people to be exposed to acetonitrile. Since EPA has established a reference concentration for acetonitrile, it would be completely contrary to generally accepted scientific principles for EPA to refuse to take into account full consideration of whether or not there would be any exposure of the human population to concentrations greater than its RfC. In conjunction with its review of the initial BP Petition to delist, EPA conducted a risk assessment and concluded there would be no exposures at levels that would be greater than the reference concentration. Accordingly, EPA should delete acetonitrile from the list of toxic chemicals in accordance with the requirements of EPCRA.

Even if this were not a direct requirement of the statute, EPA would be compelled by its own EPCRA policy to consider whether there would be exposure at levels greater than the reference concentration. At the very least, since the RfC establishes a level considered safe for lifetime exposure, exposure at that level could not be considered any more serious than low to moderately low toxicity. Accordingly, under EPA's listing/delisting policy for toxic chemicals, EPA conducts and considers an exposure assessments for chemicals with low to moderately low toxicity.

IRIS, an independent part of EPA charged with making broad determinations of risk assessment, has determined that there is a safe level for lifetime exposures to acetonitrile. Consequently, statutory requirements and EPA's own policies require that the Agency consider exposure levels to determine whether there would be any exposure at concentration levels above the one which IRIS has concluded is safe.

Based on BP's and EPA's exposure analyses, there simply will not be exposure at levels above the IRIS RfC. Accordingly, EPA should consider the exposure assessments and conclude that, because exposure will not occur at levels higher than the RfC, acetonitrile should be delisted.

VII. EPA Has Already Determined that Exposure Will Not Occur at Levels Potentially Associated with Chronic Adverse Effects.

Both the BP and EPA-sponsored exposure assessments showed that long-term exposure patterns will not exceed the RfC adopted for acetonitrile by IRIS. In response to the 1998 Petition, EPA reviewed the 1998 Exposure assessment conducted for BP and contracted to have another assessment conducted by GSC. Admittedly, the primary focus of both of these assessments was on determining the level of acute exposures for short one and twenty-four hour periods. Nevertheless, the results generated are applicable to the periods relevant for determining longer term exposure patterns. The approach taken uses the assumption that releases at the levels reported continue over 365 days per year and models ambient air concentrations on that basis. The results, as summarized in Table 3, show that exposures for the facilities that EPA modeled translate to maximum annual levels of 0.04 ppm as compared with the RfC of 0.06 ppm amount. These modeling results reflect the facilities with the highest release levels and exposure potential. By comparison, the other facilities reporting releases over the past six years would generate significantly lower exposure levels

Because exposures will occur only at levels below the RfC, these data show that exposure to the concentrations of acetonitrile likely to occur in the ambient air beyond facility fence lines will not be sufficient to cause either acute or chronic toxicity. Accordingly, EPA should delist acetonitrile from the list of Toxic Chemicals.

VIII. The Current Status of Acetonitrile as a Volatile Organic Compound (VOC) Should Not Be a Factor in the Decision to Delist Acetonitrile from the TRI.

As a separate basis for retaining acetonitrile on the TRI list of toxic chemicals, EPA stated in its 1999 Decision that "acetonitrile meets the listing criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) due to it contributing to the formation of ozone."[90/] This conclusion was based solely on EPA's determination that acetonitrile is a volatile organic compound ("VOC"). BP specifically requests EPA to reverse its position on this issue under EPCRA section 313(d)(2)(C) and not to base any denial on alleged environmental effects from ozone formation due to the status of acetonitrile as a VOC.[91/]

As discussed in more detail in the 1998 Petition, EPA has not provided — and cannot find — any legal support for its position that chemicals not otherwise toxic within the meaning of EPCRA can be retained on the list solely because the chemical is a VOC. A

90/ 64 Fed. Reg. at 10603.

91/ 64 Fed. Reg. at 10,601.

chemical cannot be listed in the first place because of environmental effects of ozone formation under EPCRA section 313(d)(2)(C). Otherwise, EPA would have a sufficient basis for adding all VOCs to the Section 313 list, but that is clearly not the case. The listing provisions of EPCRA are intended to be a door that swings both ways—that is, evidence insufficient to result in an initial listing is also insufficient to result in retention of the chemical on the list. If a chemical cannot be listed solely because it is a VOC, then it cannot be retained on the list solely because it is a VOC. In short, Congress has not authorized EPA to refuse to delist chemicals that are VOCs.

EPA decision to retain acetonitrile on the list also fails because EPA cannot demonstrate that acetonitrile actually contributes to ozone formation in the troposphere or to causing any toxic effects that may be attributable to ozone. Essentially, EPA is presuming that acetonitrile has indirect toxicity through a chain of events that leads to the formation of the toxic chemical ozone. In *Fertilizer Institute v. Browner*,[92/] the court rejected this type of reasoning for phosphoric acid on the ground that EPA was defining "toxicity" too broadly and that the adverse effects identified by EPA "are not due to any inherent property of phosphoric acid."[93/] In that case, the court appeared to distinguish VOCs from its ruling on the ground that VOCs "have toxic effects that, though indirect, are inevitable and not dependent [*sic*] on any variables or intervening causes."[94/] But this conclusion would not be true for acetonitrile. As confirmed by an EPA Office of Air & Radiation (OAR) determination that is described below, the causation chain is broken because acetonitrile does not contribute significantly to the formation of ozone. It is clear from what the court said in the case of phosphoric acid that EPA cannot simply rely on presuppositions of toxicity. The Agency must be able to sustain its listing decisions on the basis of scientifically-supported determinations of toxicity that are directly attributable to the chemical in question. EPA's presumptive labeling of acetonitrile as a VOC that contributes to ozone toxicity fails this test and must be rejected as a basis for a TRI listing decision.

Finally, even if EPA were able to sustain its legal and policy positions on the use of VOC status to retain chemicals on the TRI list, it cannot justify that action for acetonitrile. As a protective measure, BP submitted a petition to EPA's Office of Air and Radiation (OAR) to define acetonitrile as a "negligibly photoreactive chemical" under 40 C.F.R. § 51.100(s)(1) — *i.e.*, to remove acetonitrile from the definition of VOC. The petition indicates that the physical characteristics of acetonitrile meet—with a one order-of-magnitude margin of safety—the criteria used by EPA to define chemicals as negligibly photoreactive. As stated earlier,[95/] in response to the petition, OAR has agreed with BP's conclusion that acetonitrile is not

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92/ Civ. Action No. 98-1067 slip op. (D.D.C., April 15, 1999) (Exhibit S). Moreover, BP does not agree with the court's dictum in this case and contends that the same reasoning used by the court for phosphoric acid would apply in this case, especially where the toxic effects of ozone cannot be shown to result inevitably from acetonitrile emissions.

93/ Slip op. at 11.

94/ Slip op. at 11.

95/ *See* Section I.B.

photoreactive enough to contribute to any allegedly toxic effects of ozone. Specifically, OAR has indicated that "acetonitrile is probably in the lower range of VOC photochemical reactivity" where it would qualify for delisting. Consequently, OAR advised that the current VOC status of acetonitrile "should not impose a major impediment in [OPPTS's] deliberations on the [TRI delisting] petition . . . ."[96/] BP will continue to pursue the VOC petition with OAR. In the meantime, BP urges EPA to change its policy with respect to Toxic Chemicals that are VOCs or, in the alternative, not to use acetonitrile's current status as a VOC to deny the petition in light of the OAR conclusion about photochemical reactivity.

If EPA is unwilling to change this policy, then BP specifically requests that OEI coordinate action on acetonitrile with OAR. In its 1999 Decision, EPA acknowledged that "OAR's initial review of the petition indicates that acetonitrile may be a negligibly photoreactive chemical."[97/] Moreover, EPA stated:

> If OAR's initial assessment is confirmed and a rule is issued that adds acetonitrile to the list of negligibly photoreactive chemicals under 40 CFR 51.100(s)(1), then any concerns based solely on acetonitrile being listed as a VOC **would no longer be a basis for listing** acetonitrile under EPCRA section 313.[98/]

Thus, under the TRI program, EPA is looking for a final determination and ruling that acetonitrile is not photoreactive. OAR has concluded that acetonitrile is not photoreactive, but is withholding the issuance of a final rule to that effect because it wants some reassurance that acetonitrile is not a toxic chemical. BP does not believe that this is a legitimate basis on which OAR can withhold its ruling on the pending BP petition. What is most unfair, however, is that the two EPA offices risk putting BP in the classic "Catch 22" position where each office is withholding action until the other acts. Rather than put BP in this completely unfair position, BP urges the two offices to coordinate and act simultaneously to remove acetonitrile from the definition of VOC and from the TRI list of toxic chemicals. BP's respective petitions provide all of the support necessary for these simultaneous rulings.

96/ Memorandum to Mario Doa, Toxics Release Inventory Branch, EAD, from G.Tom Helms, Ozone Policy and Strategies Group, AQSSD, "Photochemical Reactivity of Acetonitrile" (Oct. 1, 1998) (Exhibit T).

97/ 64 Fed. Reg. at 10603.

98/ 64 Fed. Reg. at 10603.

IX. CONCLUSION

For the reasons set forth in this petition, EPA should conclude that acetonitrile is not a toxic chemical within the definition of Section 313(d)(2) of EPCRA and should therefore be deleted from the Section 313 list of toxic chemicals. Acetonitrile should also be excluded from the VOC definition.

Respectfully Submitted,

Dale E. Strother, DABT
BP Chemicals Inc.
1300 Wilson Blvd.
Suite 1200
Arlington, VA 22209

Robert F. Van Voorhees
Jill M. Zucker
Bryan Cave LLP
700 Thirteenth Street, N.W.
Suite 700
Washington, DC 20005-3960

Counsel to BP Chemicals Inc.

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Table 3: Top 20 TRI Acetonitrile Air Dischargers (1995-2000)

Facility	EPA Model (mg/m3, annual)		IRIS RfC	1995 Total Air Releases		1996 Total Air Releases		1997 Total Air Releases		1998 Total Air Releases		1999 Total Air Releases		2000 Total Air Releases		Six Year Averages (1995-2000)	
	1995	1996	mg/m3	Rank	Quant. (lbs)	Rank	Quant. (lbs)	Rank	Quant. (lbs)	Rank	Quant. (lbs)	Rank	Quant. (lbs)	Rank	Quant. (lbs)	Rank	Quant. (lbs)
DuPont, Memphis, TN	0.025	0.04	0.06	1	147,793	1	279,555	1	122,321	2	110,070	2	95,657	1	100,032	1	142,571
Clinton Lab, Clinton, IN			0.06	2	117,000	4	56,000	4	65,000	6	68000	4	63,000	2	71,000	3	73,333
Celanese Acetate, Rock Hill, SC			0.06	3	62,555	8	40,832	8	31,778	1	133,185	10	27,705	5	40,910	5	56,161
BF Goodrich, Henry, IL	0.007	0.008	0.06	4	59,000	2	78,532	3	66,490	5	75,441	1	118,872	4	53,526	2	75,310
Celanese Acetate, Narrows, VA	0.025	0.03	0.06	5	57,800	3	67,000	2	107,000	4	94,000	5	52,000	6	34,200	4	68,667
BP Amoco Chemical, Alvin, TX			0.06	6	54,100	16	18,300	33	4,820	34	4,866	32	4,811	34	3,013	17	14,985
Lilly Tech. Center, Indianapolis, IN	0.005	0.006	0.06	7	50,700	10	37,000	7	33,900	8	36,000	8	34,000	8	30,000	7	36,933
Equistar Chemicals, Channelview, TX			0.06	8	47,900	6	43,900	5	37,900	7	41,000	6	35,200	3	56,400	6	43,717
Dixie Chemicals, Pasadena, TX	0.08	0.02	0.06	9	38,858	9	37,140	43	2,966	---	---	---	---	---	---	10	26,321
BP Chemicals, Lima, OH			0.06	10	29,000	13	20,500	13	18,600	3	102,900	20	9,810	17	11,910	8	32,120
Merck & Co., Riverside, PA			0.06	11	27,805	18	16,584	15	16,543	12	22,218	12	20,787	49	1,342	15	17,547
Tippecanoe Labs, Lafayette, IN			0.06	12	24,200	7	42,200	6	34,000	9	34,900	7	34,800	22	7,250	9	29,558
Goodyear Tire & Rubber, Check, TX			0.06	13	23,016	11	23,016	10	23,440	10	27,557	9	27,817	10	21,930	12	24,463
Eastman Chemical Co., Kingsport, TN			0.06	14	21,700	14	19,500	11	21,300	52	1,123	98	58	54	1,000	22	10,780
Sterling Chemicals, Texas City, TX			0.06	15	20,513	17	17,523	14	17,106	13	20,597	13	19,546	13	17,421	14	19,139
Pfizer, Inc., Holland, MI			0.06	16	18,800	20	14,300	12	19,800	22	8,900	19	10,300	15	14,550	18	13,733
Lilly Del Caribe, Mayaguez, PR			0.06	17	18,500	21	14,000	27	7,580	32	4,880	26	6,780	23	7,085	26	9,804
Bristol-Myers Squibb Company, East Syracuse, NY			0.06	18	15,600	19	15,770	20	14,000	20	9,700	22	8,580	53	1,089	21	10,790
Shell Chemical, Deer Park, TX			0.06	19	15,300	15	17,523	44	2,500	11	22,400	11	26,700	7	32,500	13	19,487
DuPont Sabine River Works, Orange, TX			0.06	20	14,546	12	20,553	17	15,400	14	19,434	14	17,873	12	17,457	16	17,544
Pharmacia & Upjohn Caribe Inc., Arecibo, PR			0.06	27	10,250	5	44,250	18	15,250	35	4,215	25	6,890	44	1,527	19	13,730
Solutia, Alvin, TX			0.06	---	---	---	---	9	24,000	23	8,700	28	5,700	24	6,800	20	11,300
BP Chemicals, Port Lavaca, TX			0.06	21	11,900	28	8,264	16	16,374	19	10,033	36	3,283	27	5,105	28	9,160
Shell Norco Chemical Plant, St. Charles, LA			0.06	47	1,830	44	1,540	19	14,740	15	16,500	17	11,200	14	16,700	24	10,418